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Successful pregnancy with left ventricular assist device failure in the setting of peripartum cardiomyopathy

Aurelio Vargas, BS^a, Sabiha Armin, BS^a, and Edward Yeomans, MD^b

^aSchool of Medicine, Texas Tech University Health Sciences Center, Lubbock, Texas; ^bDepartment of Obstetrics and Gynecology, Texas Tech University Health Sciences Center, Lubbock, Texas

ABSTRACT

Pregnancy is contraindicated for women with left ventricular dysfunction due to high maternal and fetal mortality. We present a case of a pregnant 31-year-old woman with a history of heart failure due to peripartum cardiomyopathy from a previous pregnancy. She had a left ventricular assist device (LVAD) and was on warfarin due to recurrent thrombosis of her device. During her course, she had multiple cardiac complications, including thrombosis of the LVAD, which required deactivation. At 32 weeks, a cesarean section was performed due to acute decompensation, and a transthoracic echocardiogram revealed a left ventricular ejection fraction of 30% to 34%, a dilated left ventricle, and moderate global hypokinesis. This case highlights the need for coordinated care from cardiologists and maternal-fetal medicine specialists to minimize symptoms to obtain ideal outcomes for mother and infant despite LVAD deactivation.

KEYWORDS Congestive heart failure; left ventricular assist device; peripartum cardiomyopathy; pregnancy

eripartum cardiomyopathy (PPCM) is a form of dilated cardiomyopathy defined as systolic cardiac heart failure with an ejection fraction (EF) <45% in the last month of pregnancy or during the first 5 months of the postpartum period. Pregnancy is contraindicated for women with left ventricular dysfunction from prior PPCM or women with New York Heart Association (NYHA) class III/IV heart failure due to high maternal and fetal mortality. Pregnancy-related risks regarding heart failure arise from the physiological changes of a hypercoagulable state, increase in blood volume, and increase in cardiac output. The consequences of PPCM for patients are deadly, with a mortality rate of 20% to 50% from complications such as arrhythmias, cardiac thrombus, and pulmonary emboli.² For women who do not respond to maximal medical therapy, heart transplantation is the last option.

CASE DESCRIPTION

A 31-year-old woman, gravida 4 para 3 with heart failure due to PPCM from her previous pregnancy, presented after a positive pregnancy test. She had a HeartWare left ventricular assist device (LVAD) due to decreased left ventricular EF

and was on warfarin due to recurrent thrombotic complications associated with her device. She also had type 2 diabetes managed with insulin and hypertension managed with amlodipine. Throughout her pregnancy, management focused on prevention of cardiac decompensation, placental insufficiency, and obstetric complications. Her medical regimen was modified to exclude drugs with a teratogenic potential. She remained on metoprolol, furosemide, and hydralazine for her PPCM and insulin and metformin for glucose control. The patient had multiple complications, including another thrombus formation that necessitated weaning and deactivation of her LVAD, poor control of her diabetes, and noncompliance with her diuretic medication. She remained on a therapeutic dose of enoxaparin for the remainder of the pregnancy to prevent further thrombosis. At 32 weeks of gestation, she decompensated with pulmonary edema and a repeat cesarean section was performed. Postpartum, the patient's transthoracic echocardiogram revealed a dilated left ventricle with an EF of 30% to 34% and moderate global hypokinesis (Figure 1). She was restarted on guidelinedirected heart failure therapy and warfarin after delivery with

Corresponding author: Aurelio Vargas, BS, TTUHSC School of Medicine, Office of Academic Affairs, Mail Stop 8326, 3601 4th Street, Lubbock, TX 79430 (e-mail: aurelio.vargas@ttuhsc.edu)

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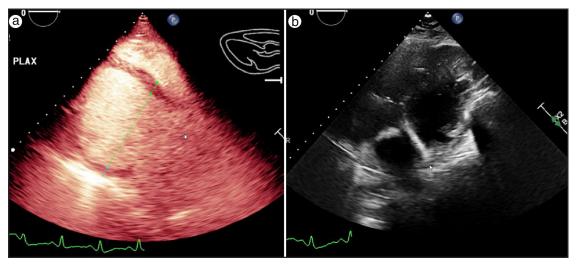


Figure 1. Transthoracic echocardiogram with contrast: (a) parasternal long axis view showing a dilated left ventricle; (b) substernal view showing LVAD canula in the left ventricular cavity.

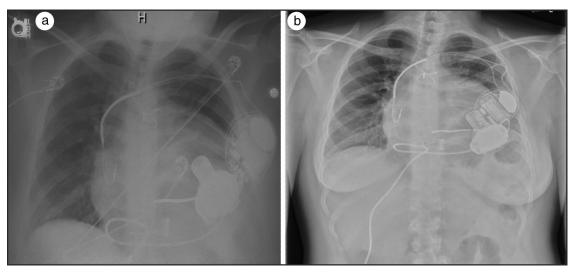


Figure 2. Chest x-ray **(a)** in the postoperative period showing no edema or intrapleural markings and **(b)** 2 months after delivery depicting no focal consolidation or edema, an enlarged cardiac silhouette, and the LVAD present and properly positioned.

no complications. LVAD management was continued by the institution where the device was originally placed (*Figure 2*).

DISCUSSION

LVAD can be utilized as bridge therapy between acute cardiac decompensation and heart transplantation. Selection criteria focus on risk stratification, symptom severity, and long-term survival outcomes in patients with heart failure. At least three cases demonstrated successful pregnancy despite LVAD deactivation; our treatment plan was similar to that in these cases, focusing on cardiac stability while reducing the teratogenic profile of the medications, as our patient wished to proceed with the pregnancy. There are no established guidelines on LVAD weaning prior to pump deactivation, though several physicians have reported favorable LVAD deactivation and explanation after LVAD insertion. This is usually done after maximized guideline-directed medical therapy (angiotensin-converting enzyme

inhibitors, beta-blockers, angiotensin receptor blockers, spironolactone, and clenbuterol) and weekly step-down LVAD deactivation through reduction of rpm, followed by serial echocardiograms to assess ejection fraction.

There is limited guidance on the management of anticoagulant therapy for pregnant woman at risk of thrombotic complications. The lowest maternal risk of thrombotic complications appears to be with the use of warfarin, but that carries the highest risk of fetal adverse events. Although lowmolecular-weight heparin offers the least risk of fetal adverse effects and is preferred in high-risk pregnancies, delaying the use of warfarin until after embryogenesis has not proven to decrease fetal risk.⁹

It is standard practice to proceed with spontaneous vaginal delivery even in pregnant patients with nonsevere cardiac dysfunction (NYHA class I or II) because cesarean section is associated with greater blood loss and higher thromboembolic and infection risk. 10 Given the patient's acute

decompensation at 32 weeks, NYHA III classification, and history of c-section, we proceeded with a repeat c-section to reduce the risk of uterine rupture and other complications. Additionally, contractions during the second stage of vaginal delivery place a high degree of hemodynamic strain, an effect we wanted to minimize. Situations in which primary c-section is indicated are labor while on oral anticoagulants, Marfan syndrome with risk of dissection, and acute heart failure. Preterm delivery is known to increase the risk of fetal hypoplastic lungs and respiratory distress, and this was seen in the patient's neonate, who required care in a neonatal intensive care unit but was successfully discharged with a good outcome.

With proper management, our patient delivered a viable preterm infant and avoided worsening cardiac function despite her deactivated LVAD due to recurrent thrombotic occlusion. As access to LVAD specialists may prove a barrier to many patients outside of large tertiary health systems, this case highlights the need for coordinated care from cardiologists and maternal-fetal medicine specialists to obtain the best outcome for mother and infant.

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